THE EFFECT OF TRAUMA AND STRAIN ON THE PRODUCTION AND AGGRAVATION OF HEART DISEASE*

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THE world is at present recovering from its greatest traumatic experience. This, perhaps, justifies a reëxamination and review of the knowledge of the effects of trauma and strain on the circulation, the more so since the war has produced new observations in this field. In recent years the subject has often been debated in the manner of a dispute with legalistic implications rather than objectively. This is inevitable where scientific proof is lacking and the possibilities of litigious heart disease are present, as in personal injury, industrial accident, and compensation cases.

Trauma is defined as an injury or wound. Such injury may vary from the damage of such shocking disasters as airplane crashes and atomic bombing to the minor entanglements of automobile fenders or the malign effects of overeating. But also this concept of trauma includes such influences as the inhalation of toxic gases and psychic injury without physical impact.

Strain, similarly, may be of short or long duration, severe, mild, usual or unusual, physical or mental.

In general, one may conclude that, in the category of harmful influences we are considering, the injury is so produced that some person, external agent, or set of circumstances to which the victim was forced to react, could be held responsible, responsible in a causative, not a legal, sense. This is in contrast with the effects of bacteria, metabolic products, or neoplastic or degenerative processes.

The literature on the subject has become extensive. One need mention only the reviews of Bright and Beck,[1] Master and his associates,[2] Boas,[3] Willius,[4] Sigler,[5] Barber,[6] Riseman and Smith,[7] Arenberg,[8] Gilbert,[9] Parsonnet and Bernstein,[10] French and Dock,[11] Blumgart,[12] Pater-

* Presented January 11, 1946 before the New York Heart Association.

son,[13] Brahdy and Kahn,[14] Fitzhugh and Hamilton,[15] and White and Glendy.[16] At least seventy-five pertinent references may be found in the past six years.

The war has drawn attention to such syndromes as the effect of blast concussion, airplane accidents, and fatal coronary artery disease in young men. Autopsy correlation has been of convincing value. None of us who has served in the Army or Navy can retain a dogmatism about the subject based on civilian experience alone.

Types of injury. It is customary to divide the types of injury to the heart due to trauma and strain into (1) Direct penetrating. (2) Direct non-penetrating. (3) Remote injury to other parts of the body or the effects of unusual physical exertion. (4) Psychic injury.

Only the first two may ordinarily injure a normal heart, but a severe general injury may rupture the heart, and strenuous physical effort may rupture a normal aortic valve or normal chordae tendineae of the mitral valve. That all of these injuries may aggravate a preëxisting cardiac disease may be admitted. Only long experience and a judicial mind can allocate the degree of damage due to the chronic process and the added disability attributable to the injury. Furthermore, the probabilities of injury and the type of injury due to trauma or strain will vary with etiologic type of the underlying heart disease. Aortic aneurysm, for example, would be liable to a rupture from strain, whereas auricular fibrillation would be more likely to ensue with mitral stenosis.

While interesting data from combat experience in this war have been added to our knowledge of *direct penetrating injury* of the heart the mechanisms are simple and obvious and will not be discussed here. Recovery with foreign bodies in the heart wall may be miraculous. The escape of the heart from injury may also be surprising. The author saw in consultation some thirty-five patients, aboard a hospital ship off Tarawa, who had been shot through the chest, with foreign bodies often lodging in the lung or mediastinum, without cardiac injury. An obvious conclusion would be that those shot through the heart or great vessels did not reach the ship.

Direct, non-penetrating, non-fatal injuries of the heart are probably commoner than previously thought by most observers. The work of Beck and others has emphasized the syndrome of the "steering wheel" injury with compression of the heart.

Arenberg noted that, in two hundred and fifty cases of non-pene-

trating chest injuries, the worst effects were in subjects with elastic chest walls, and that more damage occurred in those without rib fractures. This confirmed Sigler's[5] findings that the factors influencing the effects of trauma to the heart were (1) the flexibility of the chest, (2) the presence of underlying coronary disease, and (3) the psychoneurotic tendency or vagosympathetic imbalance of the individual.

In severe blows to the chest or in cases where the patient falls striking the thorax, it may be difficult to localize the area of the trauma. Butterworth and Poindexter,[17] however, found that the chest blows received by thirty-five Golden Gloves boxers produced no significant electrocardiographic changes in tracings recorded directly before and after the bouts. This would seem consistent with the age and normal cardiac condition of the contestants and the degree of trauma. What might happen to previously damaged hearts might well be different. We have seen one boxer, a negro, who developed tuberculous pericarditis following a precordial blow. Such a case belongs in the field of trauma and tuberculosis, rather than trauma and cardiac disease.

Certainly minor myocardial contusions may occur with relatively insignificant chest injuries, but, for proof, one must demand definite clinical signs or significant electrocardiographic changes returning to normal with recovery. What are significant electrocardiographic changes should be interpreted with extreme caution and appreciation of the normal variants of ST and T segments, especially in lead III and the chest leads, related to change of position, anoxia, and emotion. T wave inversion in leads I and II or ST segment displacements of over 2 mm. or intraventricular or A-V block are evidence of myocardial damage.

It is my opinion that failure to diagnose an occasional, slight myocardial contusion is an error more justifiable than the apprehensive opinion that minor electrocardiographic changes and effort syndrome symptoms commonly mean cardiac damage, with the resultant fixation of cardiac neurosis.

It is clear that each case must be individualized, but in the normal heart non-penetrating, non-fatal, non-shocking chest trauma may cause the following conditions: 1) Pericarditis—tuberculous, acute fibrinous, serous or hemorrhagic, or rupture of the pericardium. 2) Heart block—auriculo-ventricular, intraventicular (?). 3) Abnormal rhythms—premature beats, auricular paroxysmal tachycardia, auricular flutter, auricular fibrillation and possibly ventricular fibrillation. 4) T wave and ST

segment abnormalities. 5) Ruptured aortic valve or detachment of chordae tendineae. 6) Congestive failure from myocardial contusion; myocardial laceration with immediate or delayed rupture. 7) Angina pectoris. 8) Coronary occlusion.

Remote trauma, not producing shock, or sudden severe exertion, appears effective in these conditions: 1) Subacute bacterial endocarditis from dental extraction or from an injury which disturbs the immunity process. 2) Rheumatic fever may rarely be reactivated by an injury or surgical operation. 3) Auricular fibrillation may be induced by severe effort, injury, or electric shock. 4) Ventricular tachycardia may be produced by inhalation of chlorinated hydrocarbons—tetrachlorethylene, carbon tetrachloride, chloroform, ethyl chloride, and cyclopropane.[18] 5) The anoxia of carbon monoxide inhalation may produce myocardial necrosis and precipitate anginal or congestive failure in those with coronary artery disease. 6) Normal heart valves may be ruptured by exertion such as cranking a car, and normal chordae by an effort like rowing. Other coronary effects will be considered later.

Since these conditions have been observed and reported with adequate necropsy study, it should lead us to consider the possibility of such cardiac injury in all traumatic cases. Moreover, such patients should be kept under observation for at least two weeks with the realization that in a rare case the final effects of trauma may not appear for weeks or months, as in post-traumatic adhesive pericarditis.

The minimal evidence for injury to the heart when not otherwise explained should be one or more of these findings: 1) significant electrocardiographic changes, 2) cardiac enlargement unexplained by preexistant cardiac disease or hypertension, 3) abnormal rhythms, not including premature beats unless supported by other electrocardiographic changes, 4) pericardial friction rub or cardiac tamponade, 5) aortic diastolic or loud mitral systolic murmurs with signs of congestive failure, 6) congestive failure immediately precipitated by trauma or strain, 7) angina pectoris or myocardial infarction starting within twenty-four hours of the trauma if accompanied by distress at the time of the incident. If the injury is severe, such conditions starting at any time during convalescence can reasonably be attributed to the traumatic event.

Rarely, cardiac damage eventually fatal may not reveal itself for several hours, or even days, and in the interim the individual goes about

his business without disabling symptoms. Such has occurred in myocardial contusion leading to delayed rupture.

I have reserved for more detailed consideration the most troublesome relationship in this field, that between *coronary artery disease and trauma or strain.*

It is a peculiar fact that most everyone will agree that a man with coronary artery disease may die from sudden exertion, but there appear to be two schools of thought when doctors consider those who do not quite die from myocardial infarction accompanying or following exertion. One group believes that coronary occlusion arrives unrelated to any outside influence in the relentless process of coronary narrowing from atheroma. The other school is ever on the alert to discover in the patient's history some unusual exertion or strain which precipitated the occlusion. There is, it is true, an intermediate point of view which holds that myocardial infarction of lesser degree, subendocardial in situation, is produced by the relative ischemia of a reduced coronary flow in an atherosclerotic vessel plus added myocardial demands, the syndrome of coronary insufficiency.

Statistics have been published to show that coronary occlusion bears no relationship to effort, trauma, strain, or occupation, since no more attacks occur during the active hours of the day than would be proportionately predicted, and that many attacks occur at rest or during sleep. Other figures, while admitting the frequency of coronary occlusion during inactivity, point out that this does not preclude the possibility that a given unusual exertion may precipitate such an occlusion prematurely.

These differences are not based on a confusion of terminology since it is recognized that coronary occlusion, if slowly produced, may not cause myocardial infarction, and that conversely, infarction of the myocardium may occur from coronary insufficiency without occlusion.

The main dispute surrounds the analysis of a typical case of a patient with acute myocardial infarction with characteristic electrocardiographic changes, and survival, who had engaged in an unusual effort, or been subjected to unusual strain, directly preceding the onset of his attack. Was the attack fortuitous and coincidental or causally related to the stimulus?

Let us consider the mechanisms suggested by different authors for the production of myocardial infarct, with or without coronary occlu-

sion. Let us, furthermore, grant that underlying coronary disease is always present in these cases of myocardial infarction excepting the extremely rare direct injury to a traumatized coronary artery. In all these mechanisms a relative, or absolute, ischemia and anoxia of the heart muscle is produced.

I. *Lowered coronary blood pressure and reduced coronary flow resulting in thrombosis.* (a) Normal resting pressure for the given subject during sleep. (b) Reduced systemic blood pressure in shock. (c) Abnormal cardiac rhythms—tachycardia or bradycardia. (d) Reduced blood pressure by hemorrhage. (e) Lowered blood pressure occurring after exertion. (f) Lowered blood pressure in dehydration. (g) The reduction of cardiac output during forced expiration of effort, with closed glottis (Valsalva effect), producing coronary insufficiency.[19]

II. *Elevated coronary pressure.* (a) A rise in blood pressure from exertion, pain, or emotion results in rupture of abnormal intimal capillaries in the coronary walls. This may be favored by local anoxic effects with softening of atheromatous deposits beneath the intima. The hemorrhage may rapidly or slowly produce a subintimal projection impinging upon the opposite wall of the coronary vessel occluding the lumen, or it may rupture into the coronary vessel furnishing a raw surface for the deposition of thrombus. (b) A similar rise in blood pressure may rupture an atheromatous abscess through the intima and dislodge an atherosclerotic plaque or the contents of the abscess as material for distal occlusion of the vessel, or also produce a raw surface as a base for thrombus formation.

III. *Miscellaneous mechanisms of anoxia.* (a) Reflex coronary spasm, especially the gastro-coronary reflex occurring after meals or with hiatus hernia, gall bladder disease and other intestinal pathology. (b) The relative ischemia of effort whereby the coronary supply is inadequate to prevent myocardial necrosis in the presence of increased demands. (c) The anoxemia of anemia. (d) The anoxemia of carbon monoxide poisoning. (e) Chronic excitement of the vagus (recently disproved). (f) Inhibition of sympathetic tone as occurs in experimental animals by stimulation of the nasal mucous membrane and may occur in man by the inhalation of cold air.[9]

IV. *Other possible mechanisms.* (a) Direct mechanical injury of a coronary artery, causing spasm or hemorrhage. (b) Vitamin deficiency. (c) The effect of trauma is more severe in experimental animals under

the influence of digitalis, thyroxin, or epinephrin.

With all these possibilities one might feel indeed that any condition which disturbs the "steady state" of a person with coronary artery disease might precipitate coronary insufficiency or even coronary occlusion. How often any one of these is effective cannot be told, cannot even be suspected without much more autopsy study, nor will that answer the question of just what happens in those who survive myocardial infarction. Why then should we not be content to fall back on an appeal to coincidence, since all we can ever say is that it "might" have happened thus and so in a given case, but we cannot prove in this case that it "did" happen in this fashion?

One day last December the driver of a pick-up truck stalled his car in a snowdrift in Boston. Two men offered to push him out and one of these three men died. It seemed like a clear case of unusual physical exertion and latent coronary disease. But the man who died was the driver sitting behind the wheel of his truck and not one of the men pushing it. Was this coincidence or could we discover that the driver actually had tried to shovel the car out, that he had wrestled with the gears and steering wheel, or that the cold and the excitement of the episode were actually factors in his death? Until the whole story of such a case is known in finest detail, should we attempt a surmise? Thus it is of value to study the antecedent activities, the actual effort or strain, and the resulting condition of every patient with myocardial infarct.

In 1933 Fitzhugh and Hamilton[15] became convinced that acute coronary closure and fatal angina pectoris might be deferred in patients with known coronary disease if attention was paid to warning such patients to avoid "departures from ordinary living" or what, to borrow a term of biophysics, I have called a "steady state." They collected one hundred cases in which the following conditions appeared operative in precipitating the occlusion, or what Chief Justice Rugg called "acceleration of previously existing heart disease to a mortal end sooner than otherwise it would have come," which, incidentally, is a crucial phrase. (1) Prolonged activity and fatigue. (2) Persistence in activities that had repeatedly caused angina. (3) Travel. (4) Emotional strain. (5) Alcoholic excess. (6) Starving. (7) Medication, thyroid and bromides. (8) Sexual excess. (9) Straining at stool. (10) Gorging.

Other activities associated with myocardial infarction, including those reported by Boas are: (1) During heart failure. (2) With shock.

(3) With pulmonary embolism. (4) Acute hemorrhage. (5) Acute infection. (6) Marked tachycardia or bradycardia. (7) Hypertensive crises. (8) Chest injury. (9) Exertion—sudden, severe. (10) Allergy. (11) Cold. (12) Insulin shock and hypoglycemia. (13) Electric shock. (14) Excessive heat and humidity.

A patient in this category whom I recently examined was a sailor in his early thirties who arose one afternoon feeling well, shaved, and went to breakfast. He was suddenly stricken by the sensation of a blow in the mid-sternum as if "a man had hit me and pulled his arm away and left his fist in my chest." He survived a typical myocardial infarction. Prior to this attack he was free from all symptoms, but it appeared that he was on two weeks leave and had been drinking heavily during this time, accounting for his afternoon breakfast. In spite of what some may think this is "a departure from ordinary living" in the Navy.

Although one may remain sceptical of the causal relationship in some reported cases, the experience of World War II shows that subjecting men to exertion to which they are unaccustomed and *which they must perform in the face of severe fatigue*, has precipitated coronary failure, occlusion, and sudden death.

The reports of French and Dock[11] and of Blumgart[12] are very convincing. In the former series vigorous effort and early morning chores were the apparent cause in over fifty per cent of eight fatal cases of uncomplicated coronary disease.

In Blumgart's series of eleven cases, 30-56 years old, effort was considered the precipitant. His conclusions are notably sound: "The relationship of effort to a given attack of coronary occlusion may be certain, probable, suggestive, or improbable, or non-existent. The relation is definite if these criteria are satisfied: (1) The development and increase of cardiac symptoms such as pain or substernal distress during or immediately following unusual effort. (2) The continuation of the symptoms after cessation of effort. (3) The presence of the clinical signs and symptoms of acute myocardial infarction. (4) The development of the characteristic electrocardiographic patterns of acute anterior, posterior, or lateral wall myocardial infarction."

In 1941 Westinghouse and I[20] reported seventy-seven attacks of sudden occlusion of peripheral arteries, including the retinal arteries, by the mechanisms of embolism, thrombosis, and endarteritis in ambulatory, apparently well, individuals. These occlusions occurred four-

teen times as commonly when the person was at complete rest than when engaged in the ordinary exertions of life.

I confess that the situation in the peripheral arteries may be different from that in the heart, but it does seem as if a relatively reduced blood flow was more conducive to thrombosis than a very active one. However, a very few of these episodes occurred during severe exertion.

Willius,[4] however, has gone the whole way in one direction. Since, as he says, coronary thrombosis never occurs in a normal artery, strains in the past in the case of the afflicted individual did not produce occlusion, which occurs more often in the sedentary and at rest, then effort bears no relationship to coronary thrombosis. From a compensation viewpoint based on the emphasis on a precipitating event, he says, "The practice of genteel extortion has become so widespread as to constitute an alarming scheme of sanctioned fraudulence."

This, I feel, is a limited peacetime attitude, and too selective, but also too often it is true.

It is quite obvious that the restraining influence against soft-heartedness on our part in compensation cases is the possibility of malingering or traumatic neurosis. For that reason the symptoms of precordial pain, dizziness, faintness, sweating, sighing, and palpitation cannot be accepted alone as showing injury to the heart. Let the defense be psychiatric and not cardiologic.

But by all means let us be objective to the extent of refusing categorical answers. Let us emulate Dr. Jelley on the witness stand. He was once presented by the lawyer with the longest hypothetical question on record.[21] It was twenty thousand words and took three hours to deliver. At the end of it Dr. Jelley's answer was, "I don't know."

No matter how hard we try the legal methods may defeat our best intentions. Though we may see no relationship between a given incident and the aggravation of preëxisting cardiac disease, a lawyer may confound one by replying "then you think it is good for a man with heart disease to fall down on an icy sidewalk?"

There remains consideration of *emotional trauma.* In the main it is a good legal principle that nervous strain cannot be considered a compensable injury if the emotional trauma was of such a sort as not to be expected to harm a normal individual. Otherwise might we not all be liable for fatally irritating a man with angina?

Emotion may speed the heart and raise the blood pressure, but while

the released adrenalin acts as a coronary dilator, this beneficial effect may be negated, in the individual with coronary narrowing, by the concomitant increase in cardiac work due to the emotional discharge. Pain, induced by constriction of a limb or the cranium, will produce changes in the electrocardiogram in subjects with coronary disease.[22] The T waves may increase or decrease in amplitude, an abnormal tracing become normal, and vice versa. Occasionally this appears to be true in normal subjects.

These findings are difficult to evaluate, but show that something does take place in the coronary circuit under painful stimuli usually with increase in blood pressure, but just how this is mediated is not clear. Graybiel has shown that the startle reaction produced by firing a gun behind a normal subject can cause, in the electrocordiogram, bundle branch block, inverted T waves, and displacement of the pacemaker. One should, therefore, be reluctant to interpret too much significance into electrocardiograms purporting to indicate coronary injury from emotional stress.

The delayed appearance of myocardial infarction following a single emotional strain would seem highly unlikely. But the control of emotions may well prolong the life of a person suffering from coronary disease. One of Hamilton's patients, "a gentleman of the old school, famed for his easily aroused ire, found at the age of seventy that indulgence in bad temper caused him angina. He succeeded completely in controlling his disposition and lived for ten years in a forced good humor."

The cardiac responses effecting sudden death from exertion or emotion are not all known. Ventricular fibrillation or standstill appear to be two of them. It is hardest of all to prove the mechanism of unexpected death in apparently normal young individuals with normal hearts at necropsy.

Raab[23] has reported the autopsy findings in a young man who died during a game in whom there was a very high concentration of epinephrin bodies in the heart muscle as the only abnormal finding.

One of my pathologist friends said recently that there is no reason why a normal person could not faint to death if he were propped up during syncope. Such seems to have occurred in an exhausted soldier who returned from the battlefield, ate a heavy meal, and fell asleep in sitting position against a wall.

Finally, from a practical experience in industry what may one conclude about the *danger of employing men with latent coronary disease?* I will quote only one such report. In twenty years the Eastman Kodak Company has had only one case of a death claim for aggravation of existing heart disease. At autopsy this man was found to have a syphilitic aorta with a moderate back strain as the cause of the claim.

One hundred employees died of coronary thrombosis in the years 1921-1940. In only twelve did symptoms begin at work and in only one case was there a question of aggravation by work activity. Crain,[24] who reports this experience, states that a good preëmployment physical examination defining the disability is as good as a waiver of disability in cases of compensation.

In *summary* let me emphasize what seems necessary to further the study of this ramified subject of trauma and the heart. The desiderata in every case are these: (1) An honest history, taken as soon as feasible after the event. (2) The absence of present or impending litigation. (3) The condition of the patient prior to trauma or strain. (4) The antecedent activities of the patient for at least a week before. (5) The customary physical and emotional habits of the subject. (6) The exact degree of injury or strain. (7) The history immediately after the episode. (8) The bridging of symptoms or "intercalary period." (9) The departure of the patient from his normal equilibrium following the episode. (10) The objective evidence of cardiac injury—change in heart size or function, pericardial or endocardial variants, electrocardiograms. (11) The assessment of the neurotic component. (12) The autopsy findings or the final recovery state.

Numerically, after all, this group of cases is a small part of cardiology. I do not wish to over-accent the role of trauma or strain in the production or aggravation of heart disease, since such are the exceptional and not the usual factors. Infection is vastly more important.

But, in all cases, let me plead for an open mind, full experience, and the rule of reason. This should not be a battle of conflicting authorities in which "God fights on the side of the heaviest artillery" unless such artillery be the truth, as we know it, and not as we imagine it.

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